

TRAUMA TO THE NERVOUS SYSTEM

Hunterian Lecture delivered at the Royal College of Surgeons of England
on
29th July 1965
by

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MORE THAN TWO HUNDRED years ago, John Hunter taught that injuries to the brain by mechanical means were of three types: concussion, compression, and brain wounds with loss of substance. He emphasized that "these three kinds of injury will produce symptoms similar to each other". With typical Hunterian acumen, he emphasized the importance of concussion. "When there is depression of bone or extravasation, the symptoms of concussion are lost, though it may be at the bottom of all" (Hunter, 1841).

This statement brings out very clearly the important fact that the basic problem of head injuries is concussion. Hunter also realized what often requires reiteration, that concussion is not invariably a benign and transient form of injury to the nervous system, but rather that it is the local response of such tissues to trauma. It is disquieting to realize that our understanding of this response, its mechanics and its management have progressed very slowly in all these years.

Considerable advances have been made in recognizing and treating the surgically remediable complications of head injury such as depressed fractures, intracranial haematomas of various types, and infections. However, we have not achieved equally significant progress in understanding cerebral concussion. The management of patients with closed head injuries remains empirical and the resultant mortality, though less than 20 years ago, is high. More importantly, the quality of survival after severe head injuries could be improved. Two important limiting factors in advancing these aims are as follows: first, the lack of a suitable experimental model upon which correlations between the physical and biological phenomena of the trauma can be made in a rigorous manner. Secondly, the lack of adequate series of unselected head injury patients in whom observations have been made in a uniform manner so as to make comparison of cases and evaluation of management statistically meaningful. The reason for this has been the relative absence of *planned prospective* studies of head injuries as opposed to retrospective analyses of case records (McIver *et al.*, 1958; Lindgren, 1960; Lewin, 1953; Gurdjian and Webster, 1958).

In this lecture I shall try to trace the results of 2½ years' work on methods aimed at resolving these limitations to our understanding of the effects of trauma to the nervous system.

The experimental study

This was based on the rational approach to the mechanics of brain trauma outlined by Holbourn (1943), "that damage to the brain is a consequence, direct or indirect, of the movements, forces and deformations at each point in the brain. The movements, forces and deformations are not independent; so it is sufficient to express everything in terms of deformations. These are worked out with strict adherence to Newton's laws of motion, but with approximations to the constitution of shape of the skull and brain. Hence further advances can only come from making better approximations."

It should be realized such deformations cannot be directly measured and thus we have to use the measurement of such variables as velocity and force of impact, acceleration, and intracranial pressure, and *deduce* the mechanical behaviour of the brain assuming certain properties for its structure. To cite "acceleration-deceleration" or "compression" *per se* as the mechanism of brain concussion is scarcely more meaningful than to cite the rise in temperature in the object striking the head. These are but measurable indices monotonically related to the severity of trauma. The three pseudo-alternative "mechanisms" are scarcely better, i.e. instantaneous intracranial pressure changes; excitation of a train of pressure waves in the skull and cavitation. Fundamentally, these are merely three idealizations of the same phenomena. It is difficult to distinguish rationally among the assumptions and approximations underlying these three "mechanisms" (Shea, 1964).

Methods

Eighty monkeys were used in this study. These were naïve mature animals, unselected for sex and ranging in body weight between 3 and 6 kilograms. Each animal was examined and tested by a veterinarian and declared healthy and free from diseases, including tuberculosis, prior to experimental use. All experiments were conducted under general anaesthesia produced by intravenous Pentobarbital Sodium, 15 mgm./kilo body weight, not exceeding a total dose of 150 mgm. After surgical preparation, the animal was maintained at a lesser degree of anaesthesia, wherein voluntary movements when undisturbed were minimal, superficial reflexes and aversive responses to noxious stimuli were present. This level was sustained when necessary by repeated intravenous injections of "Brevital" (methohexital sodium), usually not exceeding 5 mgm.

Details of the surgical preparation, instrumentation and complete apparatus specifications cannot suitably be given in this lecture. These will be published separately. The following is a brief summary of the physiological and physical measurements made. A Gilson polygraph recorded arterial blood pressure from the femoral artery and cerebrospinal fluid pressure from the lumbo-thoracic C.S.F. space via unbonded strain gauge transducers. Electrocardiographic recordings were taken from

standard leads on this polygraph as well as on a Grass 8-channel E.E.G. machine on which one channel of respiration (via a pneumotachograph) and six electro-encephalographic channels could also be recorded. This E.E.G. was obtained from four to six extradural electrodes screwed into the skull.

Linear acceleration of the head was obtained from a miniature (1 gram) piezo-electric accelerometer mounted by screws directly to the skull. Instantaneous changes in intracranial pressure were measured by semi-conductor strain gauges mounted in 5 mm. trephine holes in the skull and held in place in a watertight fashion by steel screws and dental cement. Experimental head injury of varying degree was produced by an air-powered gun firing a captive piston having a mass = 0.086 slug and bearing at the striking surface a rubber tip = 1 square inch. The velocity of the piston was directly measured from a voltage generated by a magnet within the piston passing through a coil. A bonded strain gauge was incorporated in the striking tip of the piston and this enabled the direct measurement of the force of impact with the head. The output from the velocity, acceleration, force and pressure transducers was fed through suitable amplifiers into a tape recorder having a frequency response up to 10 kilocycles. The tape record of this data could then be played back at any chosen speed through an ultraviolet recording oscillograph in order to obtain a faithful visual record of the impact phenomena. Figure 1 illustrates the general arrangement of the apparatus. The contoured primate chair was specially designed to allow complete mobility in positioning the animal for impact. In the foreground and above the chair can be seen two high-speed motion cameras which allowed motion pictures at speeds around 4,000 frames per second. Motion analysis of such films against the reference grid seen in the background allowed calculation of the displacement of the head in two axes (x and y) and of the consequent tangential velocity attained by the head after each blow.

Other experiments included radiographic studies using carotid angiography by a technique which has been published (Ommaya *et al.*, 1964; Rockoff and Ommaya, 1964) and a modification of the Shelden-Pudenz-Restarski-Craig technique of a lucite calvarium for direct observation of brain movement during impact (Shelden *et al.*, 1944). Experimental cerebral concussion was defined as the loss of voluntary movement, and aversive responses to ear pinch when these had been present consistently immediately before impact. The *duration* of concussion was defined as being equivalent to the duration of the loss of such responses. This criterion was found to be much more reliable than the corneal reflex. In addition to the measured indices of piston and head velocity (feet per second), linear head acceleration ($g = 32$ feet per second²), impact force (lb.) and intracranial pressure changes (p.s.i. = lb. per square inch—1 p.s.i. = 51.7 mm. Hg) calculated values for kinetic energy ($\frac{1}{2} mv^2$) in

foot-lb. and for impulse ($= \int_{t_1}^{t_2} f dt$) in lb.-sec. were all correlated with the production of concussion as defined above. Each animal was struck a varying number of blows in different positions and under varying conditions as outlined later. However, only the first blows for each animal struck either in the midoccipital regions, or midfrontal regions, *and which did not produce fracture*, were considered for purposes of statistical analysis

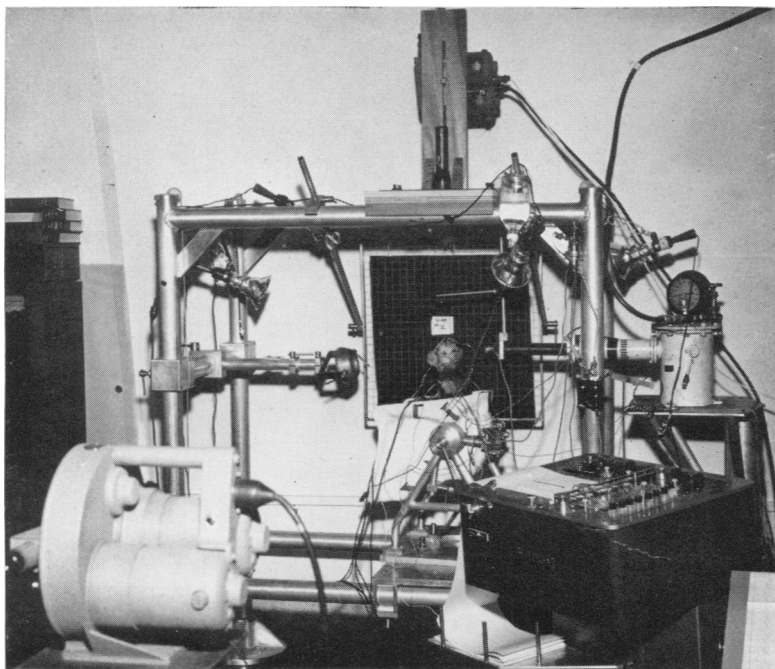


Fig. 1. Apparatus for experimental head injury in the primate. Note the compressed air gun on the right, the polygraph in the right foreground, the monkey seated in the adjustable chair, and the two high-speed cameras.

of the relation of concussion to each of the impact energy measurements described. Immediately after non-lethal experimental head injury, each monkey was allowed to regain consciousness and observed in isolation and in company with its peers for neurological and behavioural abnormalities for varying intervals of time. Any evidence of infection or discomfort was suitably treated.

At the end of each experiment the animals were sacrificed at varying intervals provided the head injury produced was not immediately fatal. All animals were sacrificed by perfusion of the major arteries with isotonic saline and 10 per cent formalin in saline under general anaesthesia pro-

duced by Pentobarbital Sodium. Post-mortem observations were made of scalp injuries and fractures, haematomas or evidence of haemorrhage, brain swelling, contusion and laceration. To this macroscopic study were added observations of blood-brain barrier breakdown as indicated by ultraviolet fluorescence of sodium fluorescein given intravenously and histological studies of routinely stained sections taken at various sites of the brain and upper cervical cord.

RESULTS

It did not appear that the actual site of impact on the head was a crucial factor in the production of concussion for occipital and frontal blows. The important factor was the efficiency of impact, i.e. whether or not the piston impacted squarely or delivered a glancing blow. The site of impact was crucial, however, for the production of skull fracture. Thus blows to the vertex produced fractures most easily, while blows to the occipital region (inion) were least likely to do so. The frontal and temporal regions were intermediate between the other regions in this regard. For purposes of this study, occipital blows were studied primarily.

A maximum of five successive blows were given to some of the animals. An interesting feature was the difficulty in producing a state of severe closed head injury with prolonged unconsciousness lasting more than a few hours in these experiments. Thus the blow (or blows) resulted in one of three things. First, no effect at all (sub-concussive blow); secondly, a concussive blow from which the animal recovered completely, usually in less than one hour. Finally, a severe concussion with or without skull fracture could occur. These animals usually died within 2-3 hours after the blow. In only 4 out of 80 monkeys (5 per cent) was there a severe concussion with survival up to 24 hours. It proved impossible to produce skull fracture *without* concussion under our experimental conditions. On the other hand, fatal concussion without skull fracture could be obtained, when death usually occurred after a short interval ranging from a few minutes to a few hours.

The usual sequence of events after a concussive blow in monkeys consisted of immediate generalized flaccid paralysis, areflexia and loss of response to noxious stimuli. In a few animals, the first effect of the blow after a brief stilling was a tonic phase of opisthotonic posturing, occasionally followed or preceded by a few clonic jerks of facial muscles or extremities. This type of response was followed by the flaccid response as described above. In the recovery phase it was quite usual to see responses to noxious stimuli return before the corneal reflex was definitely elicitable. In most cases both responsiveness and reflex actions returned together. However, the corneal reflex would often temporarily be unobtainable after initial return without concurrent changes in responsiveness to noxious stimuli, or vital signs. On the other hand, if responsiveness were abolished after initial return, reflex responses were also abolished and the animal

usually died of severe brain damage. Death in every fatal case, both with and without fracture, when observed was due to respiratory arrest and subsequent fall of blood pressure and cardiac arrest. This is in distinction to the results in the cat obtained by Denny-Brown and Russell (1941), where death was due to primary paralysis of the vasomotor centre.

A sample of the oscillographic record obtained at the time of an occipital blow is seen in Figure 2. This displays the velocity of impact (32.5 feet per second), linear acceleration of the monkey's head (205 "g"), force of

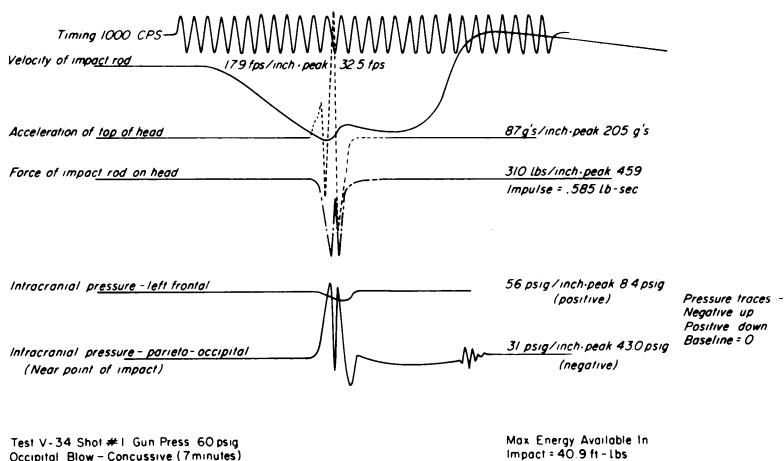


Fig. 2. Oscillographic recording of impact phenomena. Explanation in text. Events occurred from side marked by Test Number (V34).

impact (459 lb.) giving a calculated impulse = 0.585 lb. sec., and the concurrent changes in intracranial pressure at the parieto-occipital area (-43 p.s.i.) and over the left frontal lobe (+8.4 p.s.i.). This blow produced a concussion lasting for 7 minutes without fracture and with complete recovery of the animal. It is of interest to note that the high-speed motion picture of this impact confirmed an efficient impact and a calculated value of 32.6 f.p.s. was obtained for the tangential velocity of the head ($V_x = 29.3$, $V_y = 14.2$, $V_t = \sqrt{V_y^2 + V_x^2} = 32.6$). When similar data from a series of first blows given to 30 monkeys was related to the presence or absence of concussion by parametric and non-parametric statistical methods, the following results emerged. Impact velocity and kinetic energy ($\frac{1}{2}mv^2$) were significantly related to the production of concussion ($P \leq .001$) as were the linear acceleration of the head and the impulse of the blow ($P \leq .01$). However, the force of the blow, the tangential velocity of the head, and the levels of intracranial pressure change were *not* significantly related to the presence or absence of concussion. These results do not mean that any of the four significant input

measurements can be indicated as *the* mechanism of concussion, nor are they *equally* useful in their only legitimate use, i.e. as a precise index of the severity of a blow in producing concussion and other effects of experimental head injury. From a consideration of Newton's Laws of Motion, the best index would appear to be the *impulse*. Thus of the four significant indices (impact velocity, kinetic energy, impulse and acceleration) only the impulse can be usefully specified independent of the impacting masses and related to the energy input to the head. The impulse therefore provides one with an index which can be used in any experimental situation and levels of this index can be studied precisely. This point is well brought out in Tables I to V. From this data it is also evident that it is erroneous to speak of concussion as an "all-or-none" type of phenomenon related to a specific level of energy input. The tables show clearly the range of energy levels at which concussion occurs. Here, too, it would appear that impulse is the best index as indicated in the relatively smooth progression of increased percentage of monkeys concussed with increasingly higher levels of impulse. (Impulse levels below 0.12 are not concussive.) The non-significance of the tangential velocity of the head would also vitiate against the true significance of the impact velocity under our experimental conditions.

It is also clear from these results that to relate experimental cerebral concussion to "thresholds" of velocity such as the often-quoted values of impact velocities of 28 f.p.s. (Denny-Brown and Russell, 1941), 29.4 f.p.s. (Gurdjian and Webster, 1943) and 30.0 f.p.s. (White *et al.*, 1943) is essentially inadequate. There is only a rough correspondence for this index as can be seen in Table I. Thus in the range of impact velocities = 20.1 to 25.5 f.p.s. (average = 22.4) three out of nine animals were concussed (33 per cent), in the range 25.6 to 30.5 f.p.s. (average = 28.1) five out of six animals were concussed (83 per cent), but in the range 30.6 to 35.5 f.p.s. (average = 33.0) only seven out of nine (78 per cent) monkeys were concussed. Concussion in 100 per cent animals was not obtained until the impact velocity exceeded 35.6 f.p.s. However, even this figure cannot reliably be taken as a threshold for 100 per cent concussion in that it is meaningful only when the efficiency of the impact and the masses of piston and head are specified. As stated before, the same reasoning leads us to use the impulse of the blow as the best index for the energy of impact.

The dissipation of this energy in the head was measured by three indices: the velocity of the head (V_x , V_y and V_t), the acceleration of the head and the changes in intracranial pressure. The tangential head velocities for the first blow to each animal are summarized in Table V. There was *no* statistically significant relation between the changes in *intracranial pressure* and concussion, taking into account both amplitude and duration of the peak pressure at a point almost opposite the site of impact. The importance of relating the velocity of impact (V_g) to the actual velocity of the head is brought out in Table V, where it can be seen

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TABLE I
MONKEY CONCUSSION DATA
CORRELATION OF IMPACT VELOCITY RANGE WITH CONCUSSION
(Occipital or frontal blows)

Impact velocity range f.p.s.	Average velocity f.p.s.	Number of monkeys (N)	Number of monkeys concussed (Nc)	$\frac{Nc}{N} \%$
10.0 to 20.0 ..	13.9	3	0	0
20.1 to 25.5 ..	22.4	9	3	33
25.6 to 30.5 ..	28.1	6	5	83
30.6 to 35.5 ..	33.0	9	7	78
35.6 and higher ..	36.7	3	3	100

TABLE II
MONKEY CONCUSSION DATA
CORRELATION OF KINETIC ENERGY OF IMPACT WITH CONCUSSION

Kinetic energy of impact (ft.-lb.)	Average kinetic energy (ft.-lb.)	Number of monkeys (N)	Number of monkeys concussed (Nc)	$\frac{Nc}{N} \%$
0 to 11.9 ..	7.7	3	0	0
12 to 20.5 ..	17.7	6	2	33
20.6 to 30.0 ..	25.6	6	4	67
30.1 to 40.0 ..	35.0	5	3	60
40.1 and higher ..	46.2	10	9	90

TABLE III
MONKEY CONCUSSION DATA
CORRELATION OF IMPULSE WITH CONCUSSION
(Occipital or frontal blows)

Impulse range lb.-sec.	Average impulse lb.-sec.	Number of monkeys (N)	Monkeys concussed (Nc)	$\frac{Nc}{N} \%$
0.12-0.21 ..	0.181	5	1	20
0.22-0.41 ..	0.364	7	3	43
0.42-0.61 ..	0.522	12	7	58
0.62 and higher ..	0.822	7	7	100

TABLE IV
MONKEY CONCUSSION DATA
CORRELATION OF ACCELERATION RANGE WITH CONCUSSION
(Occipital or Frontal blows)

Acceleration range ("g")	Average acceleration ("g")	Number of monkeys (N)	Monkeys concussed (Nc)	$\frac{Nc}{N} \%$
40-109 ..	73	12	4	33
110-170 ..	129	5	4	80
171 and higher ..	230	6	6	100

Rise time for peak linear acceleration = 1 m. sec.

TABLE V
MONKEY CONCUSSION DATA
CORRELATION OF TANGENTIAL HEAD VELOCITY (Vt) WITH
CONCUSSION, AND RELATION OF Vt TO IMPACT VELOCITY (Vg)

Tangential head velocity Vt	Average Vt	Average Vg	Number of monkeys (N)	Number of monkeys concussed (Nc)	$\frac{Nc}{N} \%$
0-10.0 ..	2.4	34.5	1	0	0
10.1-20.0 ..	15.5	27.1	16	12	75.0
20.1-30.0 ..	24.1	28.4	22	13	69.1
30.1-40.0 ..	33.6	24.6	14	12	85.7
40.1-50.0 ..	41.2	33.7	4	4	100

that the highest average impact velocity of the piston (34.5 f.p.s.) happened to produce the lowest average head tangential velocity (2.4 f.p.s.) in one animal with no concussion. On the other hand an average impact velocity of 33.7 f.p.s. was related to an average head velocity of 40.9 f.p.s. in four animals, all of whom were concussed. The velocity of the head in the *X* and *Y* axes considered individually were not related to concussion in any better fashion, and statistically the actual velocity attained by the head was not significantly related to concussion. On the other hand the acceleration of the head was statistically significant when related to the onset of concussion under our experimental conditions.

In summary, it would appear that knowledge of the impulse of the impact and the acceleration of the head are two reliable and statistically significant indices which can be used to relate the input and dissipation of the energy of the blow to the production of experimental concussion.

Physiological effects

A concussive blow has definite effects on the respiration, blood pressure, C.S.F. pressure, electrocardiogram and electro-encephalogram. These effects are briefly summarized as follows:

1. *Respiration*

An apnoeic pause lasting for a few seconds is usually followed by a change in respiratory pattern. This can be either irregularity of rate and amplitude or both. In fatal concussions or severe concussion associated with fracture, the apnoeic pause is usually continued and the animal dies in respiratory paralysis or the return of an irregular pattern precedes a secondary apnoea and death. Artificial respiration administered as the sole therapeutic effort in such cases for as long as three hours does not reverse the situation.

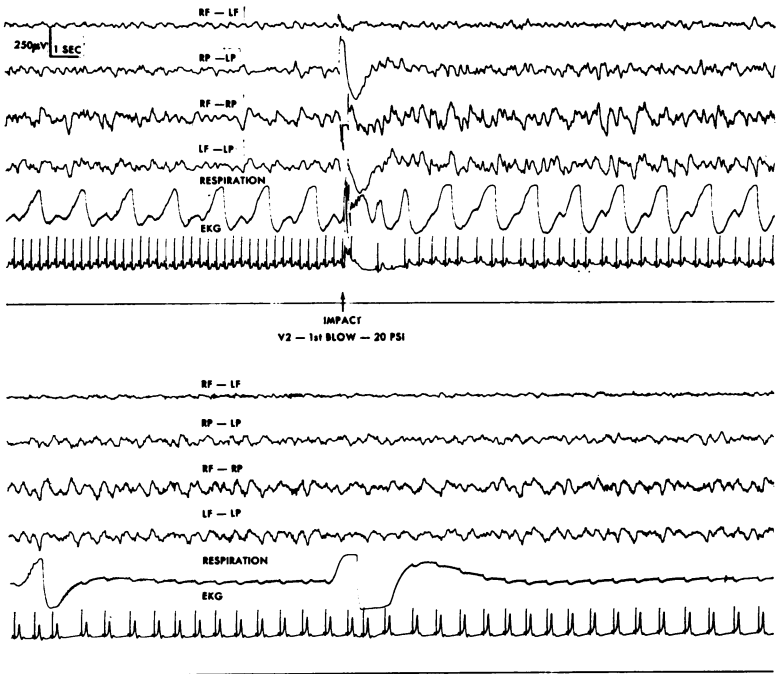
2. *Electro-encephalogram*

This was recorded in 14 animals in this series and in 18 animals in a previous series (Ommaya *et al.*, 1964). Concussion was produced in 21 of these 32 animals. The electro-encephalographic changes were fairly consistent. Thus in concussed animals an immediate onset of high-amplitude slow activity could be seen, primarily in the parietal areas bilaterally. The high amplitude of these waves would soon decrease, often before the end of concussion, although the rate would remain persistently slow. Flattening of the record as a primary event was not noted in these experiments although this was invariably present when complications such as fracture or haemorrhage occurred. Occasionally a flat slow record would replace the high-amplitude activity as a *later* phenomenon and usually after the end of concussion. In a few animals distinct spikes suggesting epileptiform activity were seen. However, these were not related to visible convulsive activity.

Figure 3 displays the E.E.G. changes in a severely concussed monkey; concurrent changes in respiration are also seen.

3. Electrocardiogram

A careful study of these changes was made in 38 animals. Table VI indicates the overall alterations in rate, pattern, or both, and shows that there is a significant relationship between the electrocardiographic abnormalities and experimental cerebral concussion. The changes are as follows: marked bradycardia, the average heart rate of 180/minute often being reduced to half; occasional dropped beats and extrasystoles



V2 - 7 MINUTES AFTER IMPACT - (20 PSI) SHOWING ONSET OF RESPIRATORY ARREST AND ASSOCIATED CHANGES IN EEG AND EKG

Fig. 3. Record of the electro-encephalogram, electrocardiogram, and respiration before, during and after experimental head injury. Upper four traces—E.E.G. Fifth trace—Respiration. Sixth trace—E.C.G. This was a fatal blow, the onset of apnoea being visible in the second set of six traces placed below the first set. Note concurrent changes in E.E.G. and E.C.G.

compounding an irregular rhythm. Premature ventricular constrictions were occasionally frequent. *T* waves were usually elevated, although occasional inverted or biphasic *T* waves were seen. The *QRS* complex often showed increased amplitude and occasionally were inverted. The *Q-T* interval was often increased and the *ST* segment depressed. Figure 4 illustrates most of these electrocardiographic changes in an animal concussed for 2½ minutes. An attempt was made to correlate the duration of such changes with that of the concussion, but this was not feasible. However, it appeared that persistence of such electrocardiographic

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abnormalities was a definitely bad prognostic sign; even if the animal regained responsiveness to pain, a persistent bradycardia, arrhythmia or pattern abnormality resulted in death of the animal within 24 hours. Not a single animal died as the result of head trauma without having these changes immediately after the impact.

4. Blood pressure

This was recorded in 20 normal animals and in four animals after both vagi were sectioned in the neck. In 15 normal animals after the first concussive blow there was invariably a sudden *fall* in blood pressure

TABLE VI
ELECTROCARDIOGRAPHIC CHANGES AFTER HEAD INJURY

Category	Number of monkeys	Change in rate	Change in pattern	Change in rate + pattern	No change	Significance
1. Concussion without fracture	22	12 (55%)	10 (45%)	8 (36%)	10 (45%)	Chi square of categories 1, 2, and 3 related individually to presence or absence of electrocardiographic changes = 9.636 P < .01
2. Concussion with fracture and death	5	5 (100%)	3 (60%)	3 (60%)	0 (0%)	
3. No concussion or fracture	11	2 (18%)	1 (9%)	0 (0%)	9 (82%)	Chi square of categories 1 + 2 compared to category 3 and related to E.C.G. change = 4.606 P < .05
Totals ..	38	19 (50%)	14 (37%)	11 (29%)	19 (50%)	

(Fig. 5). This mean pressure drop ranged from 24 to 85 mm. Hg and persisted from 30 seconds to 10 minutes before returning to pre-trauma levels. In only two animals was the initial fall of blood pressure followed by a rise of blood pressure (50 and 75 mm. Hg) above that noted before concussion. Both of these animals died as a result of the first blow about one hour after trauma. The posture of the animal during trauma did not appear to affect these responses.

With repeated *sub*-concussive blows (up to five) repeated hypotensive effects were obtained in four animals until the fifth blow in two animals, when hypertension and death ensued. The energy input of these blows was maintained constant. With repeated *concussive* blows, the second blow also produced hypotension, as did the first blow. Following a third blow, however, four monkeys displayed a rise of blood pressure by 10–15 mm. Hg after an initial hypotension lasting up to 45 seconds.

After bilateral vagotomy, concussive trauma produced a very transient fall of blood pressure by 20–30 mm. Hg for a few seconds followed by a rapid hypertension up to 60 mm. Hg above pre-concussive levels (Fig. 6).

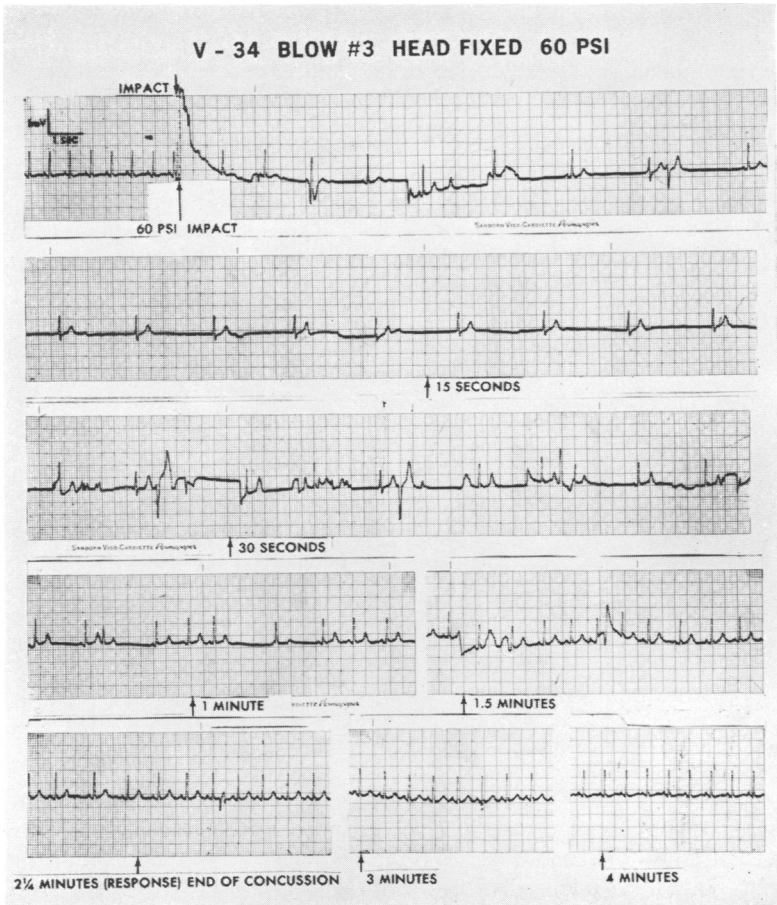


Fig. 4. The electrocardiogram before, during, and after experimental head injury. Standard limb leads. Note marked bradycardia, arrhythmia and pattern abnormalities after impact, and the return of a normal trace within 4 minutes after trauma.

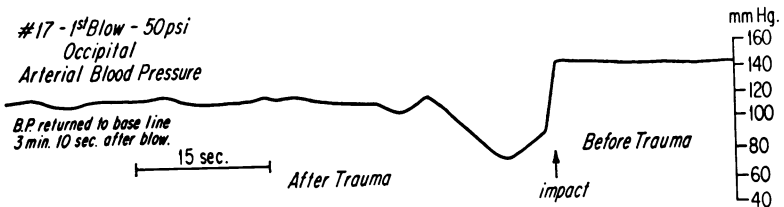


Fig. 5. The arterial blood pressure changes produced by experimental head injury. Mean pressure recording from femoral artery. Note hypotensive effect after impact.

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In one animal who had undergone two concussive blows one hour prior to vagal section, hypertension could not be produced by a concussive blow. However, repeated electrical stimulation of the femoral nerve raised the blood pressure by 20 mm. Hg (Fig. 7).

It is of interest to note that the electrocardiogram in the vagotomized monkeys remained essentially unchanged after concussive trauma, except for one animal in whom a second post-vagotomy blow produced a short period of A-V block.

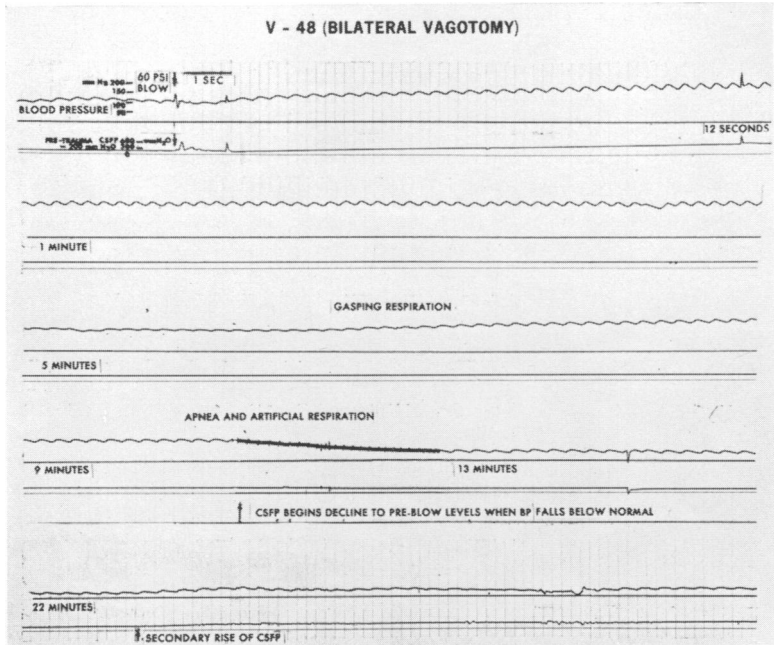


Fig. 6. Arterial blood pressure and cerebrospinal fluid pressure (C.S.F.P.) in vagotomized animal *without* prior head injury. Note preponderance of hypertension, concurrent changes in C.S.F.P., apnoea and resultant death in this animal.

There are five sets of traces (top trace = blood pressure).

5. Cerebrospinal fluid pressure

This was measured by a lumbar spinal catheter. Apart from a few transient positive to negative oscillations not exceeding 600 mm. H₂O immediately at impact, very little change was noted until minutes later, when a slight rise up to 300 mm. H₂O was noted. However, in severely concussed animals, who invariably died, immediate rise of C.S.F. pressure could be clearly seen (Fig. 6).

Radiological studies

Carotid angiograms were performed in the interval of 15 seconds to 90 minutes after trauma. When compared to control angiograms performed

prior to trauma, concussive trauma produced a marked slowing of the arterial circulation (Fig. 8). This circulatory slowing was evident in an apparently biphasic manner, i.e. marked slowing at 15 seconds after trauma with a restoration of normal clearing of the dye after 10 to 15 minutes. From 15 to 90 minutes the slowing was again re-established. Although this slowing was never evident in sub-concussive trauma and the

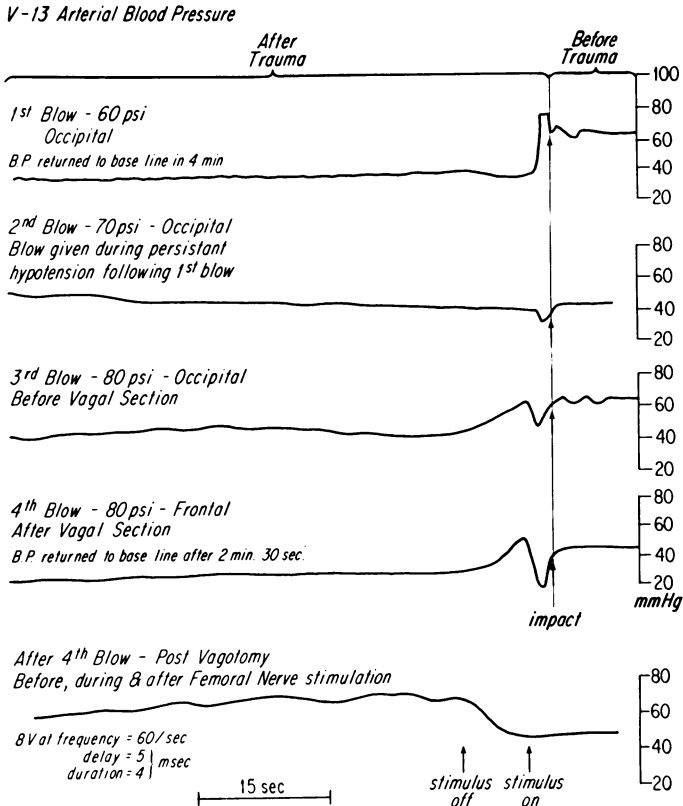


Fig. 7. Arterial blood pressure before and after bilateral vagotomy in experimental head trauma. Note marked hypotensive effect when vagotomized animal is struck after previous head injury.

production of slowing was seen in every animal concussed, the biphasic phenomenon was observed in only four experiments. These further experiments strengthened our earlier observations (Ommaya *et al.*, 1964; Rockoff and Ommaya, 1964), but much work is required to confirm this point.

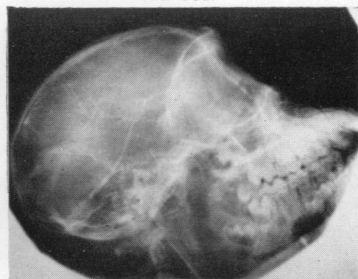
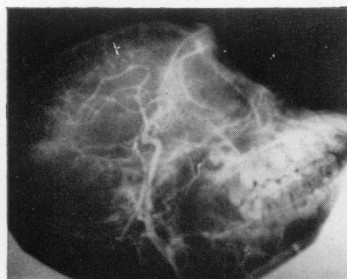
A recent radiological device which offers great potential in the understanding of impact phenomena is the Field Emission type of ultra-high-speed X-ray tube. This allows radiography at 150 kv. for extremely short

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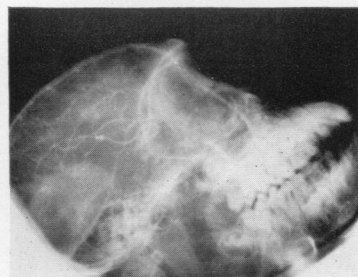
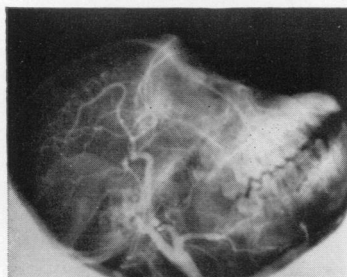
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12 Sec.

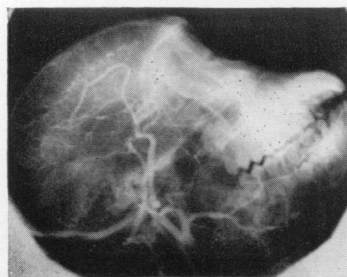
Pre-Trauma
Control
(2nd)



15 Seconds
Post-Trauma



15 Minutes
Post-Trauma



35 Minutes
Post-Trauma

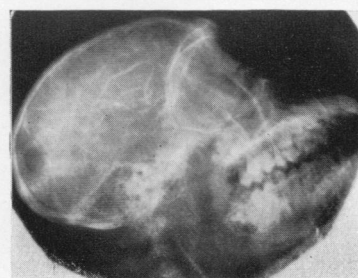
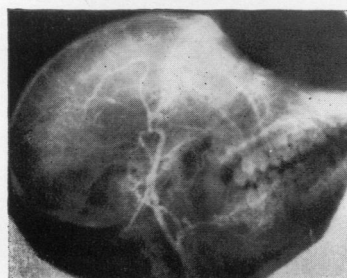


Fig. 8. Cerebral angiography after head trauma in the monkey. Note marked slowing of arterial circulation, as compared to the control angiogram (top pair). Each pair depicts the angiogram at 2 seconds and then at 12 seconds after dye injection.

exposure time (to 70 nanoseconds) and repeated exposures can be made down to intervals of 30 microseconds. This permits arrest of any movements in tissues during impact or other energy-loading situations. Preliminary results with angiography and intracerebral radio-opaque markers show that the data thus obtained is reliable. We hope quite shortly to publish full details of the actual displacements in intracranial contents

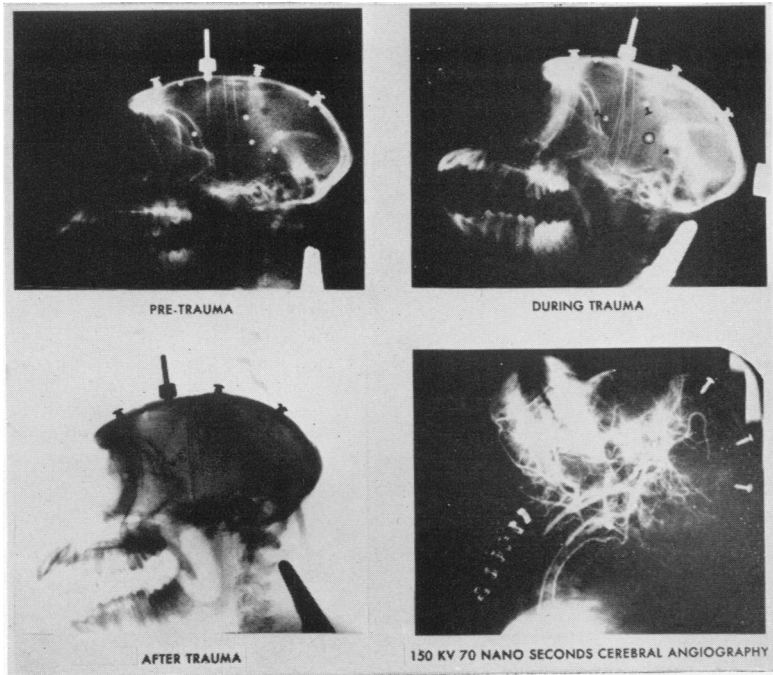


Fig. 9. Displacement of steel shot placed intracerebrally during impact to the head of a monkey, and quality of angiography is obtained by the Field Emission high-speed X-ray tube. In the frames labelled "during" and "after" trauma, a circle around the shot depicts no movement, a line drawn away from the shot shows the direction of movement of the shot, and its original position is indicated by a short line drawn at right angles to the first line. It would appear that shots "B" and "C" did not move, shots "A" and "E" moved occipitally, while shot "D" moved frontally.

during and after experimental head injury. Figure 9 illustrates our first results with this technique, showing the displacement of steel shot during and after impact, and the quality of angiographic detail obtainable.

Observations with lucite calvaria

To date, only acute experiments have been performed, in order to avoid the possibility of adhesions at the margins of dural excision introducing abnormal restrictions to brain movements. High-speed motion pictures at 3,000 to 4,000 frames per second have been made of such movements

during and after impacts to the head, and have essentially confirmed the observations of Pudenz and Shelden (1946). Thus at impact the skull moves away and the brain lags behind. The brain then swirls to follow the head, appearing to rotate on an axis, passing through the centre of gravity of the head. Maximal movements seem to be found in the fronto-parietal areas. These movements are almost identical to those predicted for the brain by Holbourn (1943). We also confirmed the marked damping of these movements by fluid by exaggerating the "sub-arachnoid space" under the lucite dome.

Behavioural studies

An attempt was made to develop an analogue to testing for amnesia in man, by training the naïve monkey in a large series of visual discriminations until immediately before head injury. The degree of loss of such learned discriminations and the rate of re-learning after experimental cerebral concussion was proposed as an index of the disruption of memory mechanisms by the trauma. The experiment could be controlled for the effect of anaesthesia, surgery and other interventions. However, the results of a preliminary testing of this analogue were not conclusive in that the post-traumatic interval before testing was too long. Further work along these lines is being pursued.

General behavioural observations of all animals recovering from trauma were made for periods up to one year. No significant physical or neurological abnormalities were seen. Post-traumatic epilepsy was never seen to occur. Isolated and colony behaviour in terms of feeding, sex, fear and aggressive-passive behaviour with peers remained unaltered. Particular attention for the appearance of any evidence of the Kluver-Bucy syndrome ("psychic blindness", oral tendencies, hypermetamorphosis for visual stimuli, loss of fear and aggression, increased sexual activity, hyperphagia) revealed no evidence for any of its manifestations.

Pathological observations

In a previous study (Ommaya *et al.*, 1964) an attempt had been made to correlate the production of experimental cerebral concussion with the leakage of intravenously administered sodium fluorescein into the brain stem and upper cervical cord. However, after further study it would appear that this is *not* a constant occurrence. Fluorescence of the cut surface of the brain stem under ultraviolet light certainly did *not* occur with non-concussive trauma, but it was also absent in many animals who had sustained definite concussion. In those concussed animals in whom fluorescence was seen, it was usually limited to a few millimetres from the surface of the brain stem rather than uniformly present over the whole surface (cf. Figures 11 to 15 in Ommaya *et al.*, 1964). The occurrence of fluorescence below the impact point, its presence in a fan-shaped manner along the posterior parasagittal areas, alongside the falx, occasionally straddling the central sulci, and the rarity of its appearance over "contre-

coup" areas were confirmed. Macroscopic observations, other than those mentioned above, were also made for evidence of fracture, clots, haemorrhage, contusions and lacerations. Animals with skull fracture were not considered in the statistical evaluation of impact-concussion relationships. There was nothing of novel merit in the observations on fracture. On the other hand the occurrence of haemorrhage and its distribution was noteworthy. First, there was not a single case of significant acute or chronic extra- or subdural haemorrhage over the convexity of the cerebral or cerebellar hemispheres in those animals *not* sustaining a fractured skull. Acute subdural haemorrhage without fracture was limited to the base, particularly around the pons and Sylvian fissures. This was in most cases associated with subarachnoid haemorrhage also in the same areas. Definite contusions and lacerations, other than those below fracture sites, were extremely unusual, and when present were usually below the impact point, around the pons, on the medial surfaces along the falx, and in the Sylvian fissures and the adjacent temporal and orbital-frontal cortex. True "contra-coup" lesions, i.e. lesions "opposite" to the point of impact, were significantly rare. Including the cases with skull fracture, only four out of 80 animals displayed such lesions. Moreover, the distribution of these "contra-coup" changes was similar to the contusional changes described above, i.e. related more to the presence of such structures as the dural and bony projections and partitions rather than to any geometric relationship to the point of impact.

Microscopic studies have to date been limited to haematoxylin and eosin preparations of paraffin sections taken from all anatomical situations in 10 animals sacrificed at intervals of 30 minutes to nine months after head injury. These sections have not revealed any changes of note. More extensive studies including Nissl, silver and myelin stains will be reported separately. A study using Marchi techniques is being undertaken under the direction of Dr. Sabina Strich, and this will be reported separately.

The experimental model for controlled head injury in the monkey described above has been developed with two intentions. First, to establish methods for prevention or protection against the effects of impact, i.e. the prophylaxis for head injuries. Secondly, to minimize these effects and prevent complications or to treat such effects and complications so that the morbidity and mortality of the experimental head injury is reduced to the minimum. The next stage would then be to establish scaling methods to apply these findings in man, both as prophylaxis for individuals in whom the hazard of trauma is always present, and in the management of patients with trauma to the nervous system. The model itself is valid inasmuch as the onset of concussion as defined is significantly related to the physical variables measured. It will also be possible to define, under identical experimental conditions, a 50 per cent and 99 per cent "lethal dosage" of energy input under a variety of conditions, which would produce fatal concussion without fracture of the skull. This would provide a tool for

testing both prophylactic and mortality reducing therapeutic techniques with a reasonable chance for success. It would appear, however, that the *morbidity* of most head injuries in man *cannot* be adequately reproduced by experimental techniques in the monkey. Thus, after repeated trauma the majority of our animals survived with *no* obvious sequelae of note. Only a few (less than 10 per cent) succumbed as a direct result of the trauma, and of these the majority had suffered compound depressed fractures.

Preliminary testing for protection against head trauma consisted of establishing the importance of movement at the cranio-spinal junction in the genesis of concussion. Thus recent experimental work by Hollister and his colleagues (1958) has indicated that stretch of the neck can produce "concussion" in the cat. Martinez and his colleagues (1963) have shown that experimental whiplash in rabbits (150 "g" linear acceleration in 20 to 120 m.sec.) can produce brain injury with surface contusions and basal haemorrhages *without* impact to the head. In a recent paper Liss (1965) has reported the death of a young swimmer who, after a racing turn involving voluntary hyperflexion, torsion and hyperextension of the head on the neck, rapidly died with agonal dyspnoea, convulsions and apnoea. Post mortem showed a congested brain, medullary oedema and petechial perivascular haemorrhages at C2-3 within the grey columns, without bony injury. In a previous study we reported the protective effect of a cervical collar against experimental concussion by head impacts in the monkey (Ommaya *et al.*, 1964). This observation was accordingly tested again and would appear to hold true. These facts have raised the very important practical point that crash helmets may, in themselves, constitute a hazard under certain conditions. Protective helmets have been designed primarily as energy absorbing or deflecting devices (Cairns and Holbourn, 1943; Lewin and Kennedy, 1956; Snively and Chichester, 1961; Gurdjian *et al.*, 1964). But by adding further weight and by shifting the centre of gravity up and forward, a heavy helmet, such as is worn by pilots and motorcyclists, increases the moment of inertia about the cervical pivots. This increases the tensile and shear stresses in the brain and cervical cord under conditions allowing acceleration and free movement of the head on the neck (indirectly, by a whiplash or flexion effect or directly by impact to the head). Existing helmet design has not taken the above into consideration, and our effort in the field of prophylaxis of cerebral concussion is to establish definite recommendations concerning the importance of these factors and ways of making provision for them.

As mentioned before, our model of experimental cerebral concussion will allow the testing of therapeutic measures to reduce the mortality in a controlled manner. We are currently planning to undertake this for hypothermia, hyperoxygenation and steroid therapy. The problems of reducing morbidity, however, cannot be suitably studied in this experimental model. For this reason clinical data from patients suffering with

head injuries is the only available source of information on which further advances can be made. Unfortunately, as pointed out by a previous lecturer in this College, the standards of accurate and uniform record keeping for cases of head injury in these times of uneasy peace very seldom reach that attained in many centres during the war (Jennett, 1961). In order to obtain statistically meaningful clinical data it was essential, therefore, to develop a system of recording, classifying and analysing such data in as uniform and precise a fashion as possible. This has been the main effort of our clinical study.

The clinical study

Existing diagnostic classifications of closed head injury are not satisfactory. Most of them are based on the definition given by Munro (1938), which add little to the facts known by John Hunter. Thus brief loss of consciousness without other signs after head injury is called concussion; a similar condition but with bloody spinal fluid is called cerebral contusion or laceration—but this term is also used to describe the patient with neurological deficits and more prolonged unconsciousness; if the C.S.F. pressure is raised the diagnosis of “cerebral oedema” is advanced. If a depressed fracture or intracranial haematoma is found, this diagnosis dominates the clinical picture. Yet it is obvious that the loss of consciousness which underlies all these diagnoses may be identical to that called concussion. Moreover, the “severity” of a head injury can have two interpretations. Thus there are the prognostic danger signals of *immediate surgical importance*, such as pyrexia, compound skull fracture, respiratory abnormality, neurological deficits, convulsions, worsening of the level of consciousness after initial improvement, rising blood pressure with falling pulse rate, and bloody C.S.F. But these surgical signposts may have no relationship to the *ultimate severity of disability* produced by head injury (Denny-Brown, 1945*b*). Thus the clinical diagnoses in common usage are at best unreliable approximations to a small part of the overall picture. The work of Symonds (1928, 1962), Ritchie Russell (1932, 1961), and Denny-Brown (1945*b*) laid the foundations for a more logical approach to the classification of closed head injuries. The intentions of our clinical study were, therefore, to develop on these foundations a system of classifying such head injuries which would have greater diagnostic precision, to establish a more precise relationship between diagnosis and prognosis as opposed to the unreliable prognostic value of existing diagnostic categories, to standardize tests for early recognition of complications, to determine the best management for these complications and for the basic problem of trauma to the nervous system, i.e. cerebral concussion.

The basic tool for clinical research is the medical record or history of the patient whose disease is being studied. The efficiency of this tool is in direct proportion to the reliability with which descriptions in one

patient can be compared to another. Moreover, to facilitate such record analysis two other requirements must be met: rapid retrieval or *access* to the pertinent data and easy comparison of similar classes of information in different patients. Previous experience in the design and use of a system of coding medical data from patients with epilepsy for machine retrieval and analysis had convinced us of the great value of such methods (Ommaya *et al.*, 1963, 1964). It was decided to apply a similar technique to the problem of head injuries. The epilepsy study was a retrospective analysis of 150 patients. In this it became clear that a prospective study with clinical data available in a more uniform manner is to be preferred. A coding system for such a prospective study in patients with head injuries has been designed and is now being tested. The clinical data is easily and rapidly put on check sheets as shown in Figure 10, by various members of the medical staff. The check sheets are then sent to a central key punch operator who transfers the data to machine punched cards. There are eight cards in the system which may be listed as follows:

1. Vital Sign Card (shown in Figure 10).
2. General Physical and Neurological Examination Card.
3. Laboratory Investigation Card.
4. Psychological Testing Card.
5. Summary Card I: Mechanism of Injury.
6. Summary Card II: Amnesia, X-ray and E.E.G. Tests.
7. Summary Card III: Therapy and Sequelae.
8. Follow-up Card.

Cards 1 to 4 contain items requiring serial observation and are coded repetitively. Cards 5 to 7 are essentially one set of cards which record items not requiring serial observation and these are completed once over the entire hospital stay of the patient. Card 8 is completed at set intervals for as long as the follow-up is carried out.

The system itself is based on a 5-point scale of levels of consciousness as follows:

Level 1. Patient is oriented in time and place and is recording on-going events, i.e. the state of normal consciousness defined operationally.

Level 2. Patient is talking and/or obeying commands but is dis-oriented and not recording on-going events.

Level 3. Patient is responding to stimuli with correct localization ("purposeful") but not obeying commands.

Level 4. Patient responds to stimuli without localization, i.e. "non-purposeful", reflex or "decerebrate" response only.

Level 5. Totally unresponsive to all stimuli.

The initial effort of this coding system is to relate the *duration* of such levels of consciousness, and the *rate* of transfer from Level 5 to 1, to the duration of post-traumatic amnesia and other neurological and behavioural sequelae of head injury in terms of the *overall* disability after

CARD I — VITAL SIGN CARD

<p>Identification Information</p> <p>1-3 <input type="text"/> <input type="text"/> <input type="text"/> Study, Card, Hosp. No.</p> <p>4-11 <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> Hosp.</p> <p>12-16 <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> Case No.</p> <p>Mo. Day Yr. Month, Day, Year</p> <p>17-20 <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> Time</p>	<p>25 Corneal Reflex</p> <p>0 No Info.</p> <p>1 Present Bilat.</p> <p>2 Absent Bilat.</p> <p>3 Absent R Only</p> <p>4 Absent L Only</p>
<p>21 Level of Consciousness</p> <p>0 No Info.</p> <p>1 Oriented</p> <p>2 Talking with Confusion</p> <p>3 Responding with Localizing</p> <p>4 Responding without Localizing</p> <p>5 Totally Unresponsive</p>	<p>Blood Pressure</p> <p>26-28 <input type="text"/> <input type="text"/> <input type="text"/> Systolic</p> <p>29-31 <input type="text"/> <input type="text"/> <input type="text"/> Diastolic</p>
<p>22 Motor Activity and Attitude</p> <p>0 No Info.</p> <p>1 Normal</p> <p>2 Quiet and Motionless</p> <p>3 Intermittent Overactivity</p> <p>4 Constant Overactivity</p> <p>5 Extensor Attitude-Const.</p> <p>6 Extensor Attitude-Intermit.</p> <p>7 Flexor Spasms</p> <p>8 Movement of Head Only</p>	<p>Pulse and Respiration</p> <p>32-34 <input type="text"/> <input type="text"/> <input type="text"/> Pulse</p> <p>35-36 <input type="text"/> <input type="text"/> <input type="text"/> Respiration</p>
<p>23-24 Pupillary Status</p> <p>00 No Info.</p> <p>01 R > L - Reactive</p> <p>02 R > L - Unreactive</p> <p>03 L > R - Reactive</p> <p>04 L > R - Unreactive</p> <p>05 Both Large - Reactive</p> <p>06 Both Large - Unreactive</p> <p>07 Both Small - Reactive</p> <p>08 Both Small - Unreactive</p> <p>09 Both Normal - Reactive</p> <p>10 Both Normal - Unreactive</p>	<p>37 Type of Respiration</p> <p>0 No Info.</p> <p>1 Regular - Normal</p> <p>2 Regular - Shallow</p> <p>3 Regular - Gasping</p> <p>4 Irregular - Cheyne-Stokes</p> <p>5 Irregular - Difficult</p>
	<p>38-41 <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> Temperature - Rectal - in °C</p>

Fig. 10. The "Vital Sign" card of the head injury coding system. Numbers within the larger rectangles refer to the punched card column numbers, while the numbers in the small rectangles are the categories within each column.

trauma. The end point will be the speed and quality of return to economic independence for each patient. Intercurrent surgical and medical complications are being carefully studied and related to this scheme. Preliminary data suggest that the system is practical, and within a few years we hope to provide answers to the problems of diagnostic and prognostic precision. This study also allows the evaluation of current management of such patients in a more rigorous manner and the statistical analysis made facile by such techniques will soon provide firm guidelines to a more rational therapy.

In the course of this study we have been able to study in detail a number of cases of very severe head injury with prolonged disturbance of consciousness. Of particular interest to us has been certain *re-integrative* phenomena displayed by such patients, as they slowly struggle towards full consciousness. By this is meant an often distorted and irregularly accelerated reproduction of ontogenetic development. The patient with a severe head injury appears to retrace in his general behaviour, as well as in his neurological pattern, his own growth and maturity. This would appear to be more clearly seen in young adults than at the two extremes of age. A few investigators (Goldstein, 1942; Wadeson, 1966) have emphasized the importance of understanding such general behaviour in terms of a physiological frame of reference related to the attempt of the personality to assert itself after disruption by trauma. Disturbances of memory are the most obvious reason for behavioural difficulties and these are often related to damage in the temporal and rhinencephalic structures (Drachman and Ommaya, 1964; Serafetinides and Falconer, 1963; Glees and Griffith, 1952; Scoville and Milner, 1957). The retrieval of lost memories appears to parallel the re-emergence of the personality, and for this reason the duration of post-traumatic amnesia is a good index of severity of brain injury (Russell and Smith, 1961). But this is not the entire story. The following case history is of interest in showing how many factors are to be considered and how much intellectual and behavioural recovery is possible even when brain damage is severe. It also depicts the re-integrative emergence of the patient's adult personality through stages of apparent "infancy" and "childhood".

National Institutes of Health Case No. 05-22-33. A 22-year-old, right-handed, married female who sustained a severe closed head injury without skull fracture, after her car collided with a truck. After 24 hours in a state of restless unconsciousness, not responding purposefully to stimuli (Level 4), increasing intracranial pressure necessitated bitemporal craniotomies (in two stages) with removal of bilateral subdural haematomas and excision of the inferior temporal gyrus on the right, and 4½ cm. of superior, middle and inferior temporal gyri on the left side as measured from the tip of the temporal pole to the vein of Labbe. This was done for pulverized brain in these regions. By the fifth day after head injury, she was at Level 3 (responding to stimuli purposefully) and in three weeks had attained Level 2 (talking, but disoriented). From this time on she exhibited slow improvement and at one month her mother described her as "as if she was six years old". Childhood habits were reasserted, e.g. rubbing side of mouth continuously, and in addition oral tendencies appeared. She masturbated frequently, occasionally smeared herself with faeces, and displayed a voracious appetite. Improvement in her behaviour was noted in a step-like increase in her speech, dressing, toilet and interest in self-appearance. Her writing was initially lettered and childlike, and as her behaviour became more adult so did the handwriting. Her parents and nurses were referred to as "teachers". She recognized her parents before her husband of six weeks, and insisted on being called by her childhood nickname. Her husband was first recognized as a boy friend and fiancé before being accepted as her husband. She would play with crayons and colour books and talked about "Winnie the Pooh". She remained in Level 2 for over two months and, when finally oriented in space and time, was found to have a post-traumatic amnesia of three months with a retrograde amnesia of only a few seconds. Her personality at this stage was that of a rather emotionally labile teenager. Pre-traumatic I.Q. had been noted at 140 (full scale Wechsler). Psychological testing after orientation was established, and three months after injury, showed results as follows: Full Scale I.Q. = 88,

Verbal I.Q. = 100, Performance I.Q. = 77, Memory Quotient = 68, M.M.P.I. was normal.

A pneumo-encephalogram done at this stage showed a uniformly dilated ventricular system, cisternal and subarachnoid spaces, with the third ventricle 1.3 cm. wide in the A.P. view. She continued to improve, and 14 months after injury was able to run her household singlehanded, but had significant bouts of depression and anxiety. Testing for memory revealed no impairment of short-term learning (recent memory) and on repeated I.Q. testing the results were as follows: Full Scale I.Q. = 117, Verbal I.Q. = 125, Performance I.Q. = 102, Memory Quotient = 112, but the M.M.P.I. showed high score on the "depression" scale.

Whether this residual personality impairment is related to the temporal lobe damage and intellectual deficit is difficult to say. However, it is of interest to note that although her P.T.A. was three months, this patient had increased her levels of consciousness very rapidly from Level 5 to Level 2 within three weeks. We are pursuing further this relation of rates of improvement in levels of consciousness to the P.T.A. and final disability after head injury, but it would certainly appear to be of some prognostic value.

Considerations of time and space do not allow a full recital of our research into diagnostic and therapeutic measures in the management of head injuries, but two techniques have proven particularly useful.

I. The use of R.I.S.A. introduced into the C.S.F. and its distribution as recorded by external scintillation scanning has proven of extreme value in the accurate diagnosis of C.S.F. rhinorrhea (Ommaya, 1964; Di Chiro *et al.*, 1964). Leakages of C.S.F. after trauma are always a potent source of meningitis and there is increasing realization that, even in the absence of frank C.S.F. leakage, meningitis may develop in patients with fractures in the anterior cranial fossa (Jefferson and Lewtas, 1963). To define such actual and potential pathways of infection, intrathecal administration of only 100 μ c of R.I.S.A. and scanning immediately over the head can usually display the site of such leakages (Fig. 11). In combination with the clinical and X-ray information, a more accurate diagnosis allows a more precise surgical repair to be performed. The use of R.I.S.A. cisternography and ventriculography to define other abnormalities of C.S.F. dynamics after head injury remains to be investigated.

II. The problem of raised intracranial pressure and its management in severe closed head injuries is often a vexed issue. The use of intravenous urea and other pressure-reducing agents is attended by certain hazards when the diagnosis is not clearly established. Thus C.S.F. pressure may be raised because of cerebral oedema, sinus thrombosis, or sinus obstruction by a fracture producing hydrocephalus without oedema. Cerebral oedema may be caused by contusions or laceration, following removal of clots, develop due to metabolic and electrolyte disturbances, or be precipitated by other cerebrovascular lesions. While investigations are pursued it is occasionally of value to monitor intracranial pressure or establish drainage of the subdural or subarachnoid space. Open techniques suffice for a few hours, but for longer periods we have suggested an indwelling subcutaneous C.S.F. reservoir (Ommaya, 1963). This is seen in Figure 12. Continuous pressure recordings during treatment with drugs such as mannitol and urea, withdrawal of C.S.F. and subdural

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fluid and introduction of drugs into these spaces is as easy as performing a hypodermic injection. Using such a device we have been able to show that the recommended dose of urea (0.5 to 1.5 Gm./kilogram body weight) for reducing intracranial pressure is perhaps excessive. With such doses the pressure is reduced to extreme *negative* values, whereas 0.25 Gm. per kilogram body weight is quite adequate in reducing high pressures to a level just below normal.

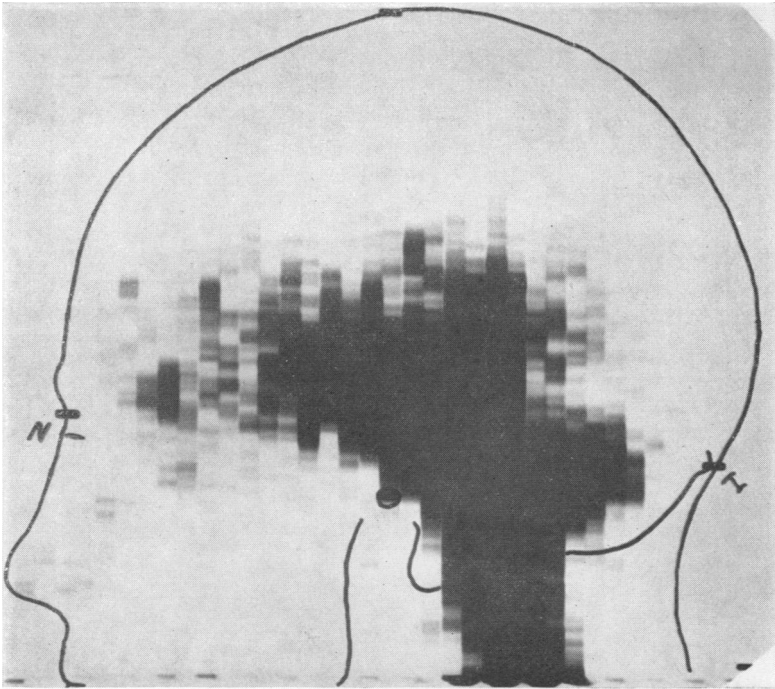


Fig. 11. R.I.S.A. cisternography in a case of C.S.F. rhinorrhea due to anterior cranial fossa fracture. Note the pathway for the leakage displayed by the positive scanning of the whole head.

In conclusion, what practical suggestions can be given for a more useful definition of concussion? The classical definition of this "violent shaking" of the central nervous system is that it is a *transient and reversible* paralysis of nervous action characterized by progressive recovery thereafter (Trotter, 1924; Denny-Brown, 1945a). But this definition does not fit all the facts. The residual post-traumatic amnesia of a true cerebral concussion is *never* a "transient and reversible" phenomenon. Experimental, clinical and pathological evidence would suggest that concussion is not an "all or none" response of the central nervous system, as it may well be in peripheral nerves. Rather it is a graded response, from the very mild, leaving *minimal* traces, to the very severe and irreversible. This

is in agreement with the views of Sir Charles Symonds (1962), who in a recent analysis of this subject stated “ concussion should not be confined to cases in which there is immediate loss of consciousness with rapid and complete recovery, but should include the many cases in which the initial symptoms are the same but with subsequent long continued disturbance of consciousness, often followed by residual symptoms ”. This statement recognizes that it is perhaps an unwarranted idealization to

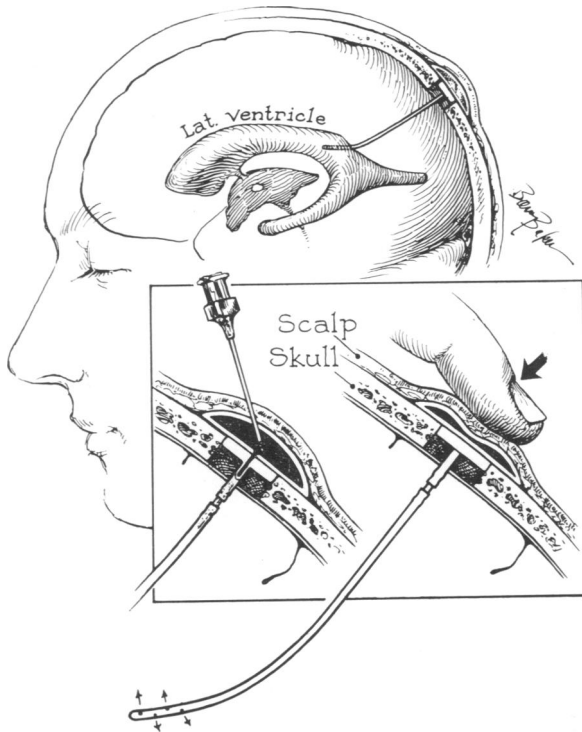


Fig. 12. A subcutaneous, chronically implantable C.S.F. reservoir useful in the management of certain cases of severe head injury.

assume that *all* the sequelae of a case of severe head injury with immediate loss of consciousness are due to undiagnosed epiphenomena or secondary complications of the head injury. It is now evident that clinical concussion can be produced both by impact to the head, as well as by bending and stretching the neck, e.g. in severe whiplash. The energy input to the brain and upper spinal cord is sudden and produces sheer and tensile stresses in the brain stem as well as in other parts of the cranial contents. Pathological evidence has revealed post-concussive degeneration of nerve fibres of spinal cord (Schmaus, 1890) and brain (Jakob, 1912; Tedeschi, 1945; Windle, 1948) as well as damaged nerve cells (Groat and Simmons,

1950; Groat *et al.*, 1945; Tedeschi, 1945; Windle and Groat, 1945; Windle *et al.*, 1944). The recent work of Strich (1961) is pertinent in that milder trauma could well produce lesser degrees of axonal damage.

It is apparent from a consideration of the evidence presented that the mechanism of concussion is not yet established. But our results support the theory of Holbourn (1943), and further work will be needed either to support or replace this theory with a better approximation. The obvious conflict of experimental data from various workers is probably related to three factors. First, the lack of uniform terminology to describe the data. Secondly, the use of quadrupeds and lower animal species in which the relation of the brain hemispheres to the brain stem and cervical cord are quite different when compared to the primates. This is illustrated schematically in Figure 13, and raises the extremely important issue of doubt in transferring concussion data from lower species to man. The similarity between the primate brain-cord geometry and that of man would perhaps make such extrapolations more permissible. Such differences may also explain the markedly *opposite* type of blood-pressure change obtained in our monkeys as compared to that in the cat and dog.

The third and possibly fundamental reason for conflicting data may also serve to explain a curious and not completely explicable fact. Thus it is known that animals with smaller brains can tolerate much higher "g" loading (acceleration) as compared to larger brained animals. Thus mice can tolerate 100 to 1,000 times the "g" level as compared to man (Kornhauser and Lawton, 1961). It would appear that this is not simply related to the mass-size ratio and that the larger brain is sensitive to much lower levels of energy as compared to smaller brains. We would suggest that this may be due to certain mechanical factors in the brain itself. Thus it has been shown that as brain weight increases from mouse through monkey to man, there is an equivalent increase of chloride, and sucrose spaces (probably related to the extracellular space) (Bourke *et al.*, 1965). Similarly, the number of neurons per cubic millimetre of brain *decrease* as the brain weight increases (Cobb, 1965). Finally, the number of glial cells per neuron (glia-to-neuron index) *increases* from small to large brains (Friede, 1954; Hawkins and Olszewski, 1957). All this would suggest that the smaller brains of lower animals tolerate injury better *because* they are more compact. The less compact, looser structure of the larger brains deforms more readily when energy is applied. These facts enable us to provide the following definition of the mechanism of concussion and attempt to relate it to this phenomenon and the other sequelae of head injury in man.

Shear or tensile deformation within the brain and cervical cord, produced by sudden impact or indirect acceleration of the head, results in dysfunction of neural elements. This dysfunction may be reversible to a degree or it may be completely irreversible in proportion to the amount of energy absorbed.

The anatomical, physiological and pathological effects in relation to the distribution of such deformation in the nervous tissues are not yet completely known, but appear to involve structures in the brain stem, hypothalamus, cortex and upper cervical cord primarily. Secondary factors (hypotension and hypertension, hypoxia, compression by fractured bone, oedema, blood or hydrocephalus, metabolic, enzymatic and endocrine changes) act invariably to enhance the effect of concussion if present after head injury, or to mimic it if it is absent. The differential vulnerability of various nerve tissues may also be related to such factors as the neurone-glial index, architectonics and the amount of extracellular space.

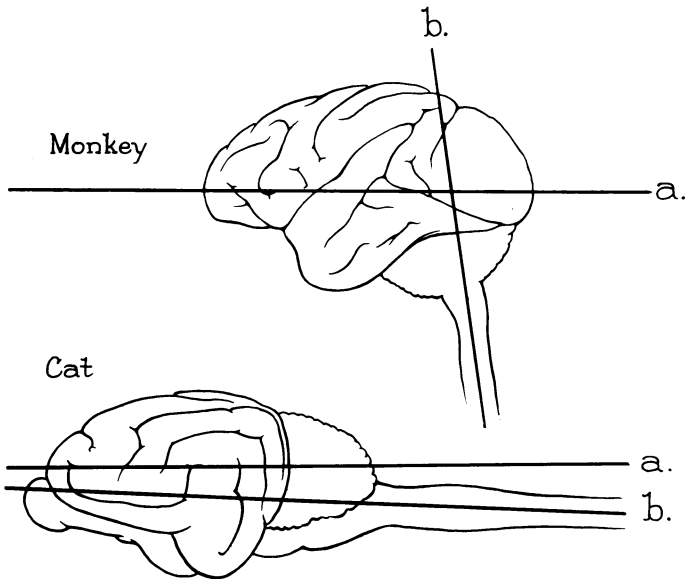


Fig. 13. The relationship between the brain hemisphere, brain stem and spinal cord axes in primates and quadrupeds, e.g. cat. This disparity is possibly the cause of variation in the responses to head injury between species. (See text for details.)

In man, the word "concussion" should be used as a general term, indicating injury to the nervous system by rapid energy loading and having as its prime index *impairment of consciousness*. This definition is made more useful if we then define consciousness in operational terms as that state of responsiveness which is characterized by maximum utilization of sensory input and motor output capabilities and with full capacity to store on-going events related to contemporary time and space (Ommaya, 1963). This latter requirement defines a method of measuring consciousness in terms of the post-traumatic amnesia.

With regard to the use of such a definition for classifying head injuries, the following plan is offered. In the present state of knowledge, it is

essential that wherever possible clinical entities related to head injury should be described rather than labelled. Terms such as comatose, contusion, brain stem lesion, etc., should be avoided. Thus all head injuries should be initially classified, when first seen, in two main categories, concussive or non-concussive. Each of these should then be qualified as falling in one of four classes, i.e. with or without fracture of the skull, and with or without complications. The complications are to be considered in two groups: (1) the local response, including vascular, parenchymal, bony, C.S.F. leakage, infection, thermoregulatory, metabolic and endocrine complications; (2) the general response. This includes complications related to the airway, chest, shock, multiple injuries, fat embolism and systemic metabolic and endocrine disturbances.

This initial diagnosis will be completed during the course of the patient's stay in hospital. A second diagnosis must then be made after a *minimum* of one year follow-up (or death) in which the total disability and length of post-traumatic amnesia are related to the initial diagnosis. By doing this consistently for a very large series of patients, using our coding system, we hope to arrive at more quantitative values for diagnosis that can be made very early after the onset of the head injury syndromes and thus guide our management and prognosis more accurately. In this effort, we will investigate particularly the role of vascular complications whose importance has been advanced by our observations on the changes in blood pressure, electrocardiogram and in the cerebral angiogram.

I will end by remarking that perhaps I have dwelt very little on the purely surgical aspects of head injuries. My reason for this is simply that the techniques of neurosurgery play a well-established but relatively minor role in less than 10 per cent of all cases. The other 90 per cent of patients from their onset, however, are automatically potential candidates for surgery until proven otherwise. It behoves us, as surgeons, to be aware, not only of the moment when surgery will save or salvage a life, but also as physicians to ensure everything possible to guarantee the *best* survival from man's oldest disease—trauma. John Hunter's advice to surgeons operating on the cranium is well worth recalling, displaying as it does that combination of surgical wisdom and utter honesty that was so much his style. "We should scalp carefully . . . and yet I own I cannot always call to mind this caution at all times when operating" (Hunter, 1841).

ACKNOWLEDGEMENTS

The work of many of my colleagues has enabled this lecture to be prepared. The past support and continuing collaboration of the following is gratefully recognized:

Mr. Richard Mahone and Mr. Arthur Hirsch of the Personnel Protection Branch, David Taylor Model Basin, U.S.N.; Dr. John Coe, Dr. T.

Krueger, Dr. Eugene Flamm and Mr. Stephen Garrell of the Branch of Surgical Neurology, National Institute of Neurological Diseases and Blindness, National Institutes of Health, have worked on various aspects at various stages. Dr. S. David Rockoff of the X-ray Department has provided radiological techniques and advice. Mrs. D. Sadowsky and Dr. James Mosimann of the Biometrics Branch, N.I.N.D.B., N.I.H., have given freely of their abilities in designing the coding system and testing the data. Dr. Igor Klatzo, Dr. Donald Tower and Dr. Maitland Baldwin have provided valuable help, criticism and advice at all stages. Accelerated development of the experimental work has been ensured by collaborative arrangements now established between the Branch of Surgical Neurology, N.I.N.D.B., N.I.H., Laboratory of Biophysics, Naval Medical Research Institute, United States Navy (Dr. David Goldman) and the David Taylor Model Basin, Personnel Protection Branch (R.M. and A.H.), with the additional financial support provided by the Bureau of Weapons, United States Navy, to whom gratitude is expressed.

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TRAUMA TO THE NERVOUS SYSTEM

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THE WORSHIPFUL COMPANY OF BARBERS

THE LORD MAYOR OF LONDON, Sir Lionel Denny, M.C., laid the Foundation Stone of the new Barber Surgeons' Hall on 6th November 1966. The new hall is in Monkwell Square, close to London Wall.

The Master of the Company, the Rev. George Turner, M.A., thanked the Lord Mayor, who is the senior past-Master of the Barbers' Company, for so graciously performing this important ceremony. The Chaplain of the Company, the Rev. B. W. Ottaway, delivered a dedicatory prayer which concluded the ceremony, after which the Master, Wardens and Court of Assistants went to the Mansion House for a glass of sherry to celebrate a very important occasion.

The Barbers' Company, which ranks No. 17 in the livery companies of the City of London, has been in existence since 1308, when the first Master was installed.

Barber Surgeons' Hall in Monkwell Street was built by Inigo Jones in 1634. It was surrounded by a herb-garden in which the Barber-Surgeons cultivated their samples. It was this garden which no doubt saved that portion of building which escaped the Great Fire of London in 1666. During the second World War the whole of Barber Surgeons' Hall was completely destroyed.

C. W.